GLOBAL VOICES

Type 1 Brugada ECG pattern in a patient with acute myocarditis: A case report

Is this a coincidental or a consequential association?

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Introduction

Brugada syndrome (BrS) remains a rare and elusive diagnosis in arrythmology. The diagnosis is based on the surface electrocardiogram (ECG), and the physiopathological mechanism is still a matter of debate.[1] Many reported cases of Brugada phenocopy (BrP) were published, which is an argument in favor of the possible inducibility of that ECG pattern in different clinical settings, but the outcome of these patients is variable according to the underlying clinical diagnosis responsible for the apparent type 1 Brugada ECG pattern.[2,3]

Case presentation

A 42-year-old male patient came to the emergency department (ED) for chest pain with symptoms of common cold and fever. Initial physical examination revealed a temperature of 38.3°C, but the rest of the vital signs were normal. There were no cardiovascular risk factors except for occasional cigarette smoking.

Surprisingly, in the 12-lead ECG, a >2 mm high takeoff coved ST-segment elevation was recorded in the right precordial leads (especially lead V_2) (Figure 1) followed by a rectilinear downsloping ST segment and a negative T wave without an isoelectric line between the T wave and the J wave (no clear r'), which is in favor of the type 1 Brugada ECG pattern.

A couple of hours later, a horizontal ST-segment elevation was recorded in the lateral and posterior (basal) leads. In addition, the right precordial leads showed a slightly different pattern (>2 mm high takeoff in lead V_2 followed by a concave downsloping ST segment and a negative T wave with a slight isoelectric line between the 2 waves) but still met the criteria of the type 1 Brugada ECG pattern.

KEY FINDINGS

- Acute myocarditis inducing a Brugada electrocardiogram pattern as a phenocopy is rare but a possible differential diagnosis for Brugada syndrome.
- The pathophysiological mechanism behind this is unknown, but we suggest that myocardial inflammation plays an important role, especially in the first hours.
- In this case, the type 1 Brugada electrocardiogram pattern is transient and has a "step-by-step" progressive resolution (despite the persistence of fever and the betablockers), which could be in favor of the depolarization theory (slow conduction due to inflammation preferentially within the right ventricular outflow tract [RVOT] myocardium).
- Is it the myocardial inflammation (but no RVOT inflammation at the cardiac MRI) or the fever (despite the ongoing improvement of the electrocardiogram under fever during the first days of hospitalization) responsible for the Brugada pattern?

Blood tests revealed a significant elevation in systemic inflammatory markers (C-reactive protein 99.2 mg/L; normal value <5 mg/L) and a significant elevation of high-sensitive troponin (highest value 4966 ng/L; normal value <38 ng/L).

Urgent coronary angiography did not show any significant coronary stenosis, thrombosis, or anomalous anatomy. Echocardiography also did not show any significant wall motion abnormalities.

The usual suspect in these cases is acute viral myocarditis. Consequently, cardiac magnetic resonance imaging performed 24 hours after admission revealed a left ventricular ejection fraction of 57% (without wall motion abnormalities) alongside a subepicardial late gadolinium enhancement, and signs of acute inflammatory edema in the inferior and lateral left ventricular walls, which is in favor of acute myocarditis (Figures 2 and 3).

KEYWORDS ECG; Brugada syndrome; Brugada phenocopy; Acute myocarditis; Case report (Heart Rhythm O^2 2024;5:834–838)

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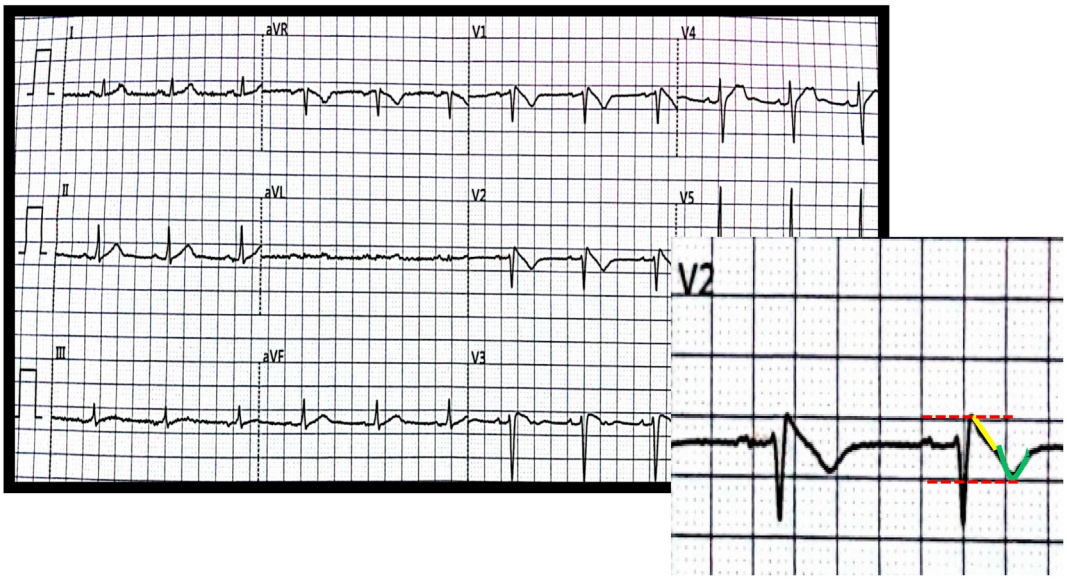

Figure 1 Twelve-lead electrocardiogram showing >2 mm high takeoff coved ST-segment elevation (*red dotted lines*) in the right precordial leads followed by a rectilinear downsloping ST segment (*yellow line*) and a negative T wave (*green line*) without an isoelectric line between the T wave and the J wave (no clear r').

A 12-lead ECG tracing was recorded on the second day showed that T waves in the right precordial leads turned positive with >2 mm high takeoff (r') and convex ST-segment elevation of 1 mm in lead V_2, favoring the type 2 Brugada ECG pattern or the "saddleback" pattern (with fragmented and low-amplitude QRS complexes in leads aVL and DI and also a negative T wave in the inferior and lateral leads) (Figure 4).

By the fifth day, there was a normalization of the right precordial leads along with the persistence of negative T waves in the lateral leads.

The patient received an angiotensin-converting enzyme inhibitor, β1-selective β-blocker, and colchicine and was scheduled for routine follow-up after discharge (on the sixth day).

One month after discharge, a follow-up 12-lead ECG (Figure 5) showed a normalization of the right precordial and lateral leads. Also, a 24-hour Holter ECG did not show any significant ventricular arrhythmias or dynamic Brugada pattern.

It is noteworthy that despite the persistence of fever (temperature > 38°C) and before receiving any treatment, the type 1 Brugada ECG pattern did not appear since the ED admission and the first day of admission (in the cardiology department).

Discussion

According to the diagnosis criteria for BrS,[1] all these arguments are in favor of BrP in a patient with acute myocarditis and not the coincidental association between the 2 pure

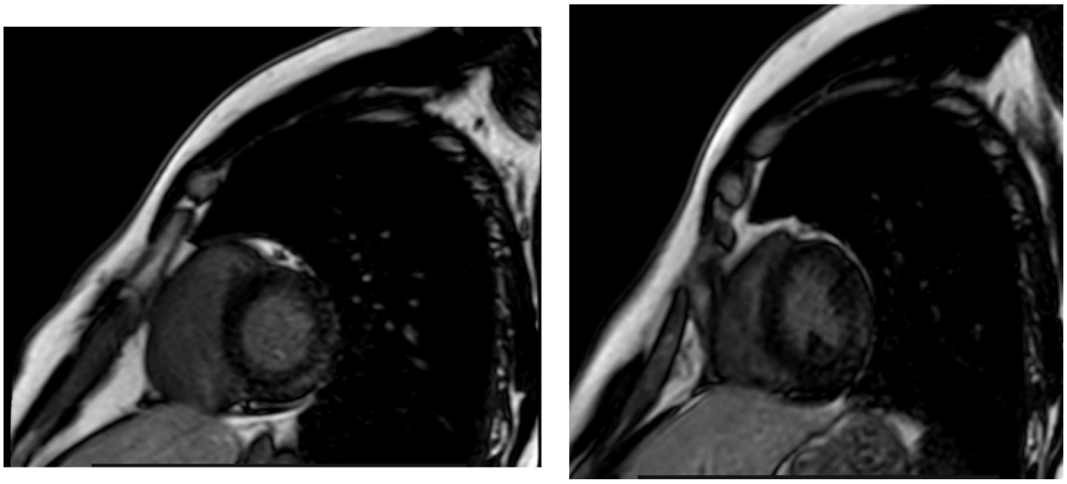

Figure 2 Short-axis view showing late gadolinium enhancement in the lateral left ventricular wall.

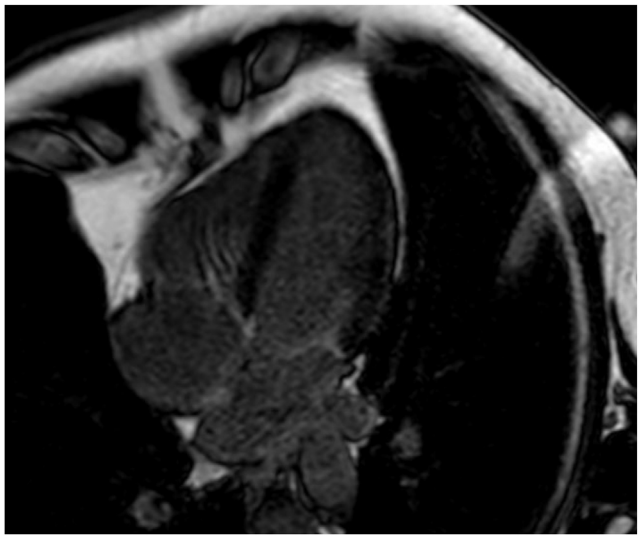

Figure 3 Four-chamber view showing late gadolinium enhancement in the lateral left ventricular wall.

cardiac diseases (BrS as a primary electrical disease and acute viral myocarditis) because of the following:

1. The low pretest probability: There were no related symptoms, no personal history of syncope or arrhythmias, and no familial history of sudden cardiac death, arrhythmias, or other congenital cardiac anomalies.
2. The presence of an identifiable acute underlying condition.
3. The resolution of the ECG pattern with myocarditis, entering remission.

The provocative pharmacological test was not conducted because of the acute phase of myocarditis (considered unsafe to do the test); the absence of an intravenous sodium channel blocker to carry out the test; the low pretest probability; resolution of the ECG pattern with myocarditis, entering remission; and the normal results of the resting ECG and 24-hour Holter ECG 1 month after the acute phase. Consequently, this case can be classified as type 1 class B BrP according to the classification of Gottschalk and Anselm.[2]

Moreover, the Shanghai score of the patient was at least 3 if we consider it a fever-induced Brugada ECG pattern without a high risk of arrhythmic events or a family history of sudden death.[4]

BrPs are regarded (at least until now) as an epiphenomenon secondary to an identifiable acquired cause rather than an original electrical abnormality. We want to try to add some explanations for some knowledge gaps throughout this real-life clinical scenario.

In terms of diagnosis, there is strong evidence that ECG patterns of BrP and BrS are visually identical and indistinguishable; therefore, systematic diagnosis criteria are highly advisable to differentiate between the 2. According to the literature, the distinction between the 2 relies mainly on the 3 aforementioned criteria: low pretest probability, underlying identifiable cause, and resolution of the ECG pattern after remission of the cause.[3,5]

In terms of mechanism, many speculations are discussed in the literature mainly about metabolic imbalances but all assumptions are channeled into the 2 main hypotheses for BrS: the depolarization and repolarization models.[6] "Myocarditis-induced BrP" is, to our knowledge, very rare; the explanations behind the ECG pattern are mainly the result of myocardial inflammation and a possible ion channel current imbalance.

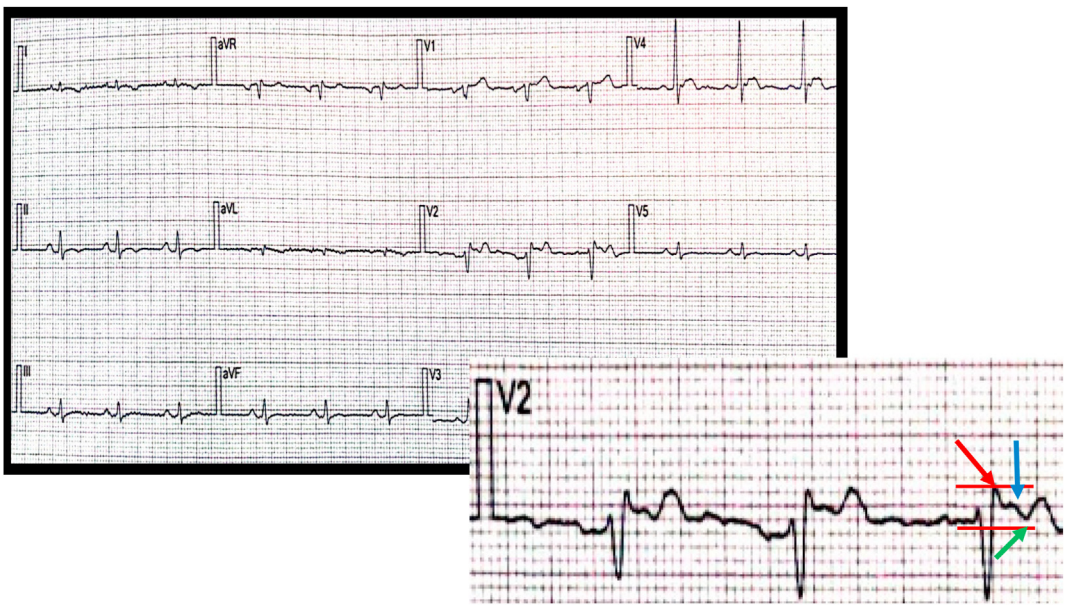

Figure 4 Twelve-lead electrocardiogram (ECG) showing that T waves in the right precordial leads turned positive (*green arrow*) with >2 mm high takeoff (r', *red arrow*) and convex ST-segment elevation of 1 mm in lead V_2 (*blue arrow*), favoring the type 2 Brugada ECG pattern or the "saddleback" pattern. Note the negative T waves in the lateral leads.

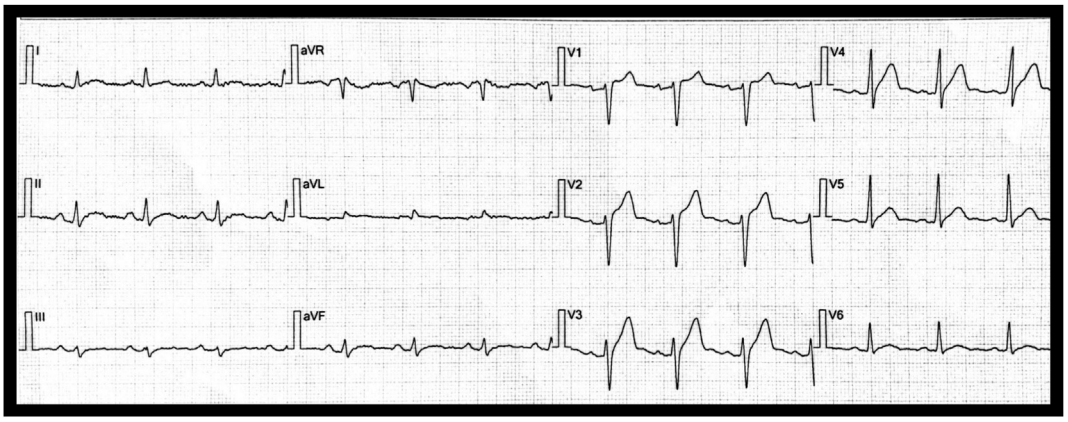

Figure 5 One month later, a 12-lead electrocardiogram showing complete normalization of the right precordial and lateral leads.

Nevertheless, the absence of cardiac magnetic resonance imaging signs of right ventricular outflow tract inflammation made us wonder: Is it the fever or the myocardial inflammation responsible for the Brugada ECG pattern? Because fever in the setting of pericardial or myocardial inflammation can provoke a type 1 Brugada ECG pattern in patients without any genetic predisposition.[7,8]

What is interesting about this case is the rapid appearance of the type 1 Brugada ECG pattern even before ST-segment elevation in the lateral and posterior leads (Figures 1 and 4) and progressive "step-by-step" resolution of the pattern from a typical type 1 Brugada ECG pattern in the right precordial leads to a different kind of ST-segment elevation couple of hours later ("less typical" type 1 Brugada ECG pattern), then a type 2 Brugada ECG pattern with positive T waves, and finally "normalization."

This is somehow favoring the depolarization theory more than the former hypothesis because of the possible progressive resolution of the conduction block along with the gradual decline in myocardial inflammation[9] (even with the administration of β-blockers, which can further slow the sodium inward current) as well as the ongoing improvement in the ECG pattern in the right precordial leads despite the persistence of fever during the first days of hospitalization (which induce the decay of the inward sodium current).

In terms of prognosis, it is unclear whether patients with BrP have a high risk of sudden death, but since the underlying cause of the phenocopy plays an important role in the pathophysiological mechanism,[6] we believe that it also has a critical role in the outcome of these patients.

Limitations

Unfortunately, the major limitation of this case is that the provocative drug test was not conducted for the previously mentioned reasons.

First, we do not know the exact sites of the placement of the right precordial leads (leads V_1 and V_2) in the first 2 ECG recordings because they were placed outside our department but we believe that these leads are at the third intercostal spaces just like in the rest of the recordings.

Second, fever may induce "artificially" a Brugada ECG pattern (just like an overdose of the sodium channel blocker) without any genetic predisposition and in a low pretest probability clinical setting (but as mentioned earlier, the type 1 Brugada ECG pattern did not appear since the ED admission and the first day of hospitalization).[6]

Conclusion

To our knowledge, BrP cases in patients with myocarditis are very rare and we believe that this case is peculiar because of the presentation and the in-hospital outcome. We suggest a more proactive approach to search for the Brugada type 1 ECG pattern in patients with acute myocarditis and to know the predisposing factors that provoke its appearance on the surface ECG to learn more about BrS.

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Ethics Statement: This case report follows the CARE guidelines.

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